WHAT IS CLAIMED IS:

1. A method for screening compounds to identify antineoplastic agents, which comprises testing said compounds for an ability to inhibit SXR trans activation of *mdr1* gene transcription.

2. A method of decreasing multidrug resistance in a cell or cells which comprises inhibiting the ability of SXR to trans activate *mdr1* gene transcription.

3. The method of claim 2, which further comprises contacting the cell or cells with an SXR antagonist, wherein the antagonist inhibits SXR trans activation of *mdr1* gene transcription.

4. The method of claim 3 wherein the contact occurs *in vivo*.

5. A method for the treatment or prophylaxis of abnormal cell proliferation in a mammal which comprises administering to such mammal an effective amount of an SXR antagonist, wherein the SXR antagonist decreases the level of *mdr1* gene transcription in the tumor cells.

6. The method of claim 5 wherein the SXR antagonist prevents displacement of an SXR corepressor from SXR.

7. The method of claim 5 wherein the SXR antagonist prevents binding of an SXR ligand to the SXR ligand binding domain.

8. The method of claim 5 wherein the SXR antagonist prevents inhibits interaction between SXR and an SXR coactivator.

9. The method of claim 5 wherein the SXR antagonist is cytotoxic to the cells of the tumor.

10. A method for treating a neoplastic disorder in a mammal which comprises administering to the mammal an antineoplastic effective amount of a cytotoxic agent and inhibiting clearance or breakdown of said cytotoxic agent by inhibiting SXR-mediated transactivation of *mdr1*.

11. A method of screening compounds for an ability to inhibit trans activation of transcription of an SXR target gene by SXR which comprises determining whether the presence of one or more of said compounds in an assay comprising SXR and said target gene inhibits transcription of said target gene as compared to transcription of said target gene in the absence of said one or more compounds.

12. The method of claim 11 wherein said assay comprises an SXR ligand.

13. The method of claim 12 wherein said ligand is a drug.

14. The method of claim 11 wherein said target gene is *mdr1*.

15. The method of claim 11 wherein said method is performed in vitro, in vivo or in cells.

16. The method of claim 11 which comprises:
a) adding an SXR ligand to cells;
b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of said ligand;
c) adding one or more of said compounds to the cells of step (a) or to cells to which SXR ligand is added;
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c); and
e) determining whether said one or more of said compounds inhibited the increase in activity or the increase in synthesis of the molecule.

17. The method of claim 16 wherein said molecule is P-glycoprotein.

18. The method of claim 16 wherein said molecule is a gene product of a reporter gene.

19. The method of claim 18 wherein expression of the reporter gene is regulated by the functional association of the ligand binding domain of SXR with an SXR coactivator.

20. The method of claim 19 wherein the SXR coactivator is selected from the group consisting of SRC1, ACTR, GRIP, PBP, a mimetic peptide which is a coactivator of SXR and a peptide fragment which is a coactivator of SXR.

21. The method of claim 19 wherein expression of the reporter gene is increased by the functional association of the ligand binding domain of SXR with an SXR coactivator.

22. An in vitro method of claim 11 which comprises:
a) mixing SXR and an SXR target gene to form a mixture;
b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of a ligand to said mixture;
c) adding one or more of said compounds to the mixture of step (a);
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c); and
e) determining whether said one or more of said compounds inhibited the increase in activity or the increase in synthesis of the molecule.

23. The method of claim 22 wherein said target gene is *mdr1*.

24. The method of claim 22 wherein said molecule is P-glycoprotein.

25. The method of claim 22 wherein said ligand is a drug.

26. A method of screening compounds for a putative antineoplastic agent which comprises determining whether the presence of one or more of said compounds in an assay comprising

SXR and a target gene of SXR inhibits transcription of said target gene as compared to transcription of said target gene in the absence of said one or more compounds.

27. The method of claim 26 wherein said assay comprises an SXR ligand.

28. The method of claim 27 wherein said ligand is a drug.

29. The method of claim 26 wherein said target gene is *mdr1*.

30. The method of claim 26 wherein said method is performed in vitro, in vivo or in cells.

31. The method of claim 26, which comprises:
a) adding an SXR ligand to cells;
b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of said ligand;
c) adding one or more compounds of said compounds to the cells of step (a) or to cells to which the nuclear ligand is added;
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c);
e) determining whether said one or more compounds inhibited the increase in activity or the increase in synthesis;
wherein a compound or compounds which inhibit said increase in activity or said increase in synthesis of said molecule are putative antineoplastic agents.

32. The method of claim 31 wherein said molecule is P-glycoprotein.

33. The method of claim 31 wherein said molecule is a gene product of a reporter gene.

34. The method of claim 33 wherein expression of the reporter gene is regulated by the functional association of the ligand binding domain of SXR with an SXR coactivator.

35. The method of claim 24 wherein the SXR coactivator is selected from the group consisting of SRC1, ACTR, GRIP, PBP, a mimetic peptide which is a coactivator of SXR and a peptide fragment which is a coactivator of SXR.

36. The method of claim 34 wherein expression of the reporter gene is increased by the functional association of the ligand binding domain of SXR with an SXR coactivator.

37. An in vitro method of claim 26 which comprises:
a) mixing SXR and an SXR target gene to form a mixture;
b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of a ligand to said mixture;
c) adding one or more of said compounds to the mixture of step (a);
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c); and
e) determining whether said one or more of said compounds inhibited the increase in activity or the increase in synthesis of the molecule.

38. The method of claim 37 wherein said target gene is *mdr1*.

39. The method of claim 37 wherein said molecule is P-glycoprotein.

40. The method of claim 37 wherein said ligand is a drug.

41. A method to screen compounds for a putative antineoplastic agent, comprising:
a) adding an SXR ligand to cells;
b) measuring an activity which is decreased or an amount of a molecule the synthesis of which is decreased by addition of said ligand;
c) adding one or more of said compounds to the cells of step (a) or to cells to which SXR ligand is added;
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c);

e) determining whether said one or more compounds inhibited the decrease in activity or the decrease in synthesis;

wherein a compound or compounds which inhibit said decrease in activity or said decrease in synthesis of said molecule are putative antineoplastic agents.

42. The method of claim 41 wherein said molecule is a gene product of a reporter gene.

43. The method of claim 41 wherein said molecule is P-glycoprotein.

44. The method of claim 41 which further comprises administering one of said compounds which inhibits said increase in activity or said increase in synthesis of said molecule to tumor cells and determining if said compound has a cytotoxic effect on the tumor cells.

45. The method of claim 41 which further comprises administering one of said compounds which inhibits said decrease in activity or said decrease in synthesis of said molecule to tumor cells and determining if said compound has a cytotoxic effect on the tumor cells.

46. A method for screening compounds as putative candidates for an ability to decrease catabolism of a drug in a cell or to decrease the ability of a cell to pump said drug out of said cell, said method comprising the steps of determining whether the presence of one or more of said compounds in an assay comprising SXR and said target gene inhibits transcription of said target gene as compared to transcription of said target gene in the absence of said one or more compounds, wherein a compound which inhibits transcription of said target gene is a candidate for decreasing catabolism of a drug or decreasing the ability of a cell to pump said drug out of said cell.

47. The method of claim 46 wherein said assay comprises an SXR ligand.

48. The method of claim 46 wherein said ligand is said drug.

49. The method of claim 46 wherein said target gene is *mdr1*.

50. The method of claim 46 wherein said method is performed in vitro, in vivo or in cells.

51. The method of claim 46 which comprises:
a) adding an SXR ligand to cells;
b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of said ligand;
c) adding one or more of said compounds to the cells of step (a) or to cells to which SXR ligand is added;
d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c); and
e) determining whether said one or more of said compounds inhibited the increase in activity or the increase in synthesis of the molecule.

52. The method of claim 51 wherein said molecule is P-glycoprotein.

53. The method of claim 51 wherein said molecule is a gene product of a reporter gene.

54. The method of claim 53 wherein expression of the reporter gene is regulated by the functional association of the ligand binding domain of SXR with an SXR coactivator.

55. The method of claim 54 wherein the SXR coactivator is selected from the group consisting of SRC1, ACTR, GRIP, PBP, a mimetic peptide which is a coactivator of SXR and a peptide fragment which is a coactivator of SXR.

56. The method of claim 54 wherein expression of the reporter gene is increased by the functional association of the ligand binding domain of SXR with an SXR coactivator.

57. An in vitro method of claim 46 which comprises:

a) mixing SXR and an SXR-target gene to form a mixture;

b) measuring an activity which is increased or an amount of a molecule the synthesis of which is increased by addition of a ligand to said mixture;

c) adding one or more of said compounds to the mixture of step (a);

d) measuring an activity or amount of a molecule as in step (b) for said cells of step (c); and

e) determining whether said one or more of said compounds inhibited the increase in activity or the increase in synthesis of the molecule.

58. The method of claim 57 wherein said target gene is *mdr1*.

59. The method of claim 57 wherein said molecule is P-glycoprotein.

60. The method of claim 57 wherein said ligand is a drug.

61. A method of drug chemotherapy which comprises coadministering a drug and an agent that modulates the activity or expression of SXR.

62. A method of claim 61 which comprises coadministering a drug and an agent that downregulates the activity or expression of SXR.

63. A method of claim 61 which comprises coadministering a drug and an agent that upregulates the activity or expression of SXR.

64. A method of increasing the effectiveness of a drug which comprises coadministering said drug with an agent that modulates the actions of SXR.

65. A method of claim 61 wherein said agent is an SXR antagonist.

66. A method of claim 61 wherein said agent is an SXR agonist.

67. A method of inhibiting drug metabolism in a patient receiving treatment with said drug, which method comprises administering to said patient an effective amount of an SXR inhibitor.

68. A process for making a therapeutic composition which comprises the steps of:
a) screening compounds for an ability to inhibit SXR activity or to inhibit transcription or translation of SXR;
b) determining which of said compounds inhibit SXR activity or inhibit transcription or translation of SXR;
c) selecting a compound which was determined to inhibit SXR activity or to inhibit transcription or translation of SXR;
d) obtaining a therapeutically effective amount of said compound selected according to step (c); and
e) combining a therapeutically effective amount of the selected compound with one or more pharmaceutically acceptable excipients to form a therapeutic composition.

69. The method of claim 68 wherein said screening comprises the steps of claim 11.

70. The method of claim 68 wherein said screening comprises the steps of claim 26.

71. A therapeutic composition made by the process of claim 68.

72. A method of inhibiting drug resistance by administering an effective amount of a therapeutic composition of claim 71 which modulates SXR activity or SXR expression.

73. A method for selecting a compound for use for treating a pathological condition in a mammal wherein said compound is selected by:

a) preparing a system comprising a ligand binding domain of SXR and an SXR target gene wherein an interaction between said ligand binding domain of SXR and said target gene produces a detectable signal;

b) measuring said detectable signal of said system in step (a);

c) adding a compound to a system of step (e);

d) measuring a signal of said system of step (c); and

e) selecting a compound wherein said signal of step (d) is less than said signal of step (b).

74. The method of claim 73 wherein said interaction is a direct interaction.

75. The method of claim 73 wherein said interaction is an indirect interaction.

76. The method of claim 73 wherein said pathological condition is a cancer.

77. The method of claim 73 wherein said target gene is *mdr1*.

78. The method of claim 73 wherein said detectable signal is *mdr1* RNA.

79. The method of claim 73 wherein said detectable signal is P-glycoprotein.

80. The method of claim 73 wherein said system comprises a cell.

81. The method of claim 80 wherein said cell comprises a vector comprising said target gene.

82. The method of claim 73 wherein said system comprises a ligand that binds to said ligand binding domain of SXR under physiological conditions.

83. The method of claim 82 wherein said ligand is a drug or drug candidate.

84. The method of claim 83 wherein said drug or drug candidate is to treat cancer.

85. The method of claim 73 wherein said system comprises components of a two-hybrid assay.

86. The method claim 85 wherein said system comprises a vector encoding an SXR ligand binding domain fused to a peptide which activates said SXR ligand binding domain.

87. The method of claim 85 wherein said peptide is VP16.

88. The method of claim 85 wherein said system comprises a vector encoding a signal generating enzyme.

89. The method of claim 73 wherein said method is performed *in vitro*.

90. The method of claim 89 wherein said system comprises a ligand binding domain of SXR and said target gene.

91. The method of claim 90 wherein said target gene is *mdr1*.

92. A compound for treating a pathological condition in a mammal wherein said compound is selected by the method of claim 73.

93. A pharmaceutical composition comprising said compound of claim 92.